Application No.: 09/509648 Docket No.: JJJ-P01-569

AMENDMENTS TO THE CLAIMS

In the claims:

For the convenience of the Examiner, all claims under consideration, whether or not amended, are presented below. Please enter the following amended claims:

- 1. (Currently amended) A method for potentiating morphogen activity, comprising administering to a mammal a composition, the composition comprising a molecule that overcomes morphogen inhibition, thereby potentiating morphogen activity.
- (Currently amended) A method for promoting neuronal cell growth, comprising
 administering to a mammal a composition, the composition comprising a molecule that
 overcomes morphogen inhibition, thereby so as to potentiate growth-promoting effects of
 endogenous morphogens thereby promoting neuronal cell growth.
- 3. (Currently amended) A method for treating a disorder characterized by neuronal cell loss, comprising administering to a mammal a composition, the composition comprising a molecule that overcomes morphogen inhibition, thereby so as to potentiate growth-promoting effects of endogenous morphogens, thereby promoting growth of a neuronal cell and treating a disorder characterized by neuronal cell loss.
- 4. (Currently amended) A method for treating a neurodegenerative disorder, comprising administering to a mammal a composition, the composition comprising a molecule that overcomes morphogen inhibition, thereby so as to potentiate morphogen activity, thereby stimulating neuronal growth by morphogens, to treat treating a neurodegenerative disorder.
- 5. (Previously presented) The method of claim 1, wherein said morphogen activity is endogenous.
- 6. (**Previously presented**) The method of claim 1, wherein said morphogen activity is the result of an exogenously provided morphogen.

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 (Previously presented) The method of claim 4, wherein said composition further comprises a morphogen.

- 8. (Previously presented) The method of claim 3 or 4, wherein said disorder is Alzheimer's disease, Parkinson's disease, Huntington's disease, senile dementia, alcohol-induced dementia, or stroke.
- 9. (Previously presented) The method of claim 1, 2, 3 or 4, wherein said molecule that overcomes morphogen inhibition is a cytokine antagonist, a retinoid antagonist, or a protein kinase A inhibitor.
- 10. (Currently amended) The method of claim 9, wherein said the molecule is a cytokine antagonist which is a neuropoetic cytokine antagonist.
- 11. (Previously presented) The method of claim 10, wherein said neuropoetic cytokine antagonist is an LIF (Leukemia-Inhibitory Factor) antagonist or a CNTF (Ciliary Neurotrophic Factor) antagonist.
- 12. (Currently amended) The method of claim 11, wherein said neuropoetic cytokine antagonist is a LIF (Leukemia-Inhibitory Factor) antagonist which is a monoclonal antibody to the a gp130 protein.

13 – 15 (**Cancelled**)

- 16. (Previously presented) The method of claim 7, wherein said morphogen comprises an amino acid sequence selected from a sequence:
 - (a) having at least 70% homology with the C-terminal seven-cysteine skeleton of human OP-1 (Osteogenic Protein 1), residues 330-431 of SEQ ID NO: 2;
 - (b) having greater than 60% amino acid sequence identity with said C-terminal seven-cysteine skeleton of human OP-1;
 - (c) defined by Generic Sequence 7, SEQ ID NO: 4;
 - (d) defined by Generic Sequence 8, SEQ ID NO: 5;
 - (e) defined by Generic Sequence 9, SEQ ID NO: 6;
 - (f) defined by Generic Sequence 10, SEQ ID NO: 7; or

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(g) defined by OPX, SEQ ID NO: 3.

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- 17. (Previously presented) The method of claim 7, wherein said morphogen is human OP-1 (Osteogenic Protein 1), mouse OP-1, human OP-2 (Osteogenic Protein 2), mouse OP-2, 60A, GDF-1 (Growth/Differentiation Factor-1), BMP2A (Bone Morphogenesis Protein 2A), BMP2B (Bone Morphogenesis Protein 2B), DPP (Decapentaplegic), Vgl, Vgr-1 (Vg1-related sequence), BMP3 (Bone Morphogenesis Protein 3), BMP5 (Bone Morphogenesis Protein 5), or BMP6 (Bone Morphogenesis Protein 6).
- 18. (Previously presented) The method of claim 7, wherein said morphogen is OP-1.
- 19. (Previously presented) The method of claim 1, wherein the molecule binds an endogenous ligand for a cytokine receptor or a retinoid receptor.

20-21. (Cancelled)

- 22. (Currently amended) The method of claim 19, wherein said the molecule which binds an endogenous ligand for a retinoid receptor is a retinoic acid receptor.
- 23. (Currently amended) The method of claim 19, wherein said the molecule which binds an endogenous ligand for a retinoid receptor is a retinoid X receptor.
- 23. (Cancelled)
- 24. (Previously presented) The method of claim 1, wherein the molecule is a cAMP-dependent messenger pathway inhibitor.
- 25. (Previously presented) The method of claim 24, wherein said cAMP-dependent messenger pathway inhibitor comprises a protein kinase A inhibitor.
- 26. (Previously presented) The method of claim 25, wherein said protein kinase A inhibitor is (2-p- bromocynnamylaminoethyl)-5-isoquinolinesulfonamide, an enantiomer of dibutyryl cAMP, or an enantiomer of cAMP.

27 – 32. (Cancelled)